TREADMILL STRESS TEST USING BODY SURFACE MAPPING
IN CORONARY ARTERY DISEASE
— The Clinical Significance of ST Depression —

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In this study we assessed in 27 patients with coronary artery disease whether the size of the ST-depression area, which was measured by body surface maps recorded before and after treadmill exercise, could be a useful indicator for evaluating coronary artery disease (CAD) quantitatively. The patients were divided into 3 groups on the basis of the findings of the left ventriculograms: patients with anterior asynergy (n = 6), those with inferior asynergy (n = 6) and those with no asynergy (n = 15). Coronary arteriograms were evaluated according to Pujadas, and ΣGRADE, as an index of the severity of CAD, was developed by adding the grade numbers of the 4 main coronary stems (right coronary artery, main trunk of the left coronary artery, left anterior descending artery and left circumflex artery). Patients with inferior asynergy and with no asynergy have ST-depression areas in proportion to their ΣGRADE (r = 0.845, p < 0.001), whereas none of the patients with anterior asynergy showed ST-depression areas regardless of their ΣGRADE. Of 6 patients who had anterior asynergy, 5 (83%) had pathologic Q waves in the left anterior chest leads. These findings emphasize the clinical value of the ST-depression area for the quantitative and non-invasive diagnosis of CAD, especially in patients without pathologic Q waves in the left anterior chest leads.

SIGNIFICANT ST depression provoked by treadmill exercise is a well established index for prediction of coronary artery disease in male patients with chest pain. However, few studies have used body surface maps for evaluating the cardiac electrical changes caused by the treadmill test. In this study we recorded body surface maps before and after treadmill exercise and tried to correlate the size of the area which showed significant ST depression with the findings of coronary arteriograms and left ventriculograms.

MATERIALS AND METHODS
This is a study of 27 male patients, admitted to our hospital complaining of chest pain, who underwent a treadmill exercise test using body surface mapping in conjunction with coronary arteriography and left ventriculography. Twelve normal volunteers underwent the exercise test only. Those with unstable angina, valvular dysfunction, cardiomyopathy or congenital heart

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Key Words:
Body surface mapping
Treadmill stress test
Coronary artery disease
Exercise-induced ST depression

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disease were excluded from the study. Patients with variant angina pectoris were also excluded. None of the 27 patients had electrocardiographic evidence of intraventricular conduction defects, and none had been treated with beta-blocking drugs or digitalis preparations.

The exercise was performed using a motor-driven treadmill according to the Sheffield protocol modified by the insertion of a 3-minute intermediate stage (stage 5') during which 3.8 mph and a 14% grade were used (Table I). The lead-CM5 ECG was monitored continuously and cuff blood pressure measurement was obtained each minute throughout the test. Exercise was continued until exhaustion, severe dyspnea or progressive angina occurred, or ST depression or elevation of more than 3 mm was seen in asymptomatic patients.

Body surface maps were recorded using the HPM-5100 Heart Potential Mapper System (Chunichi Denshi Company, Nagoya), which was developed by Yamada. The localization of the lead points and the procedure used for the data sampling and processing were described in detail in his report. For the construction of body surface maps 87 lead points were distributed on the anterior (59 points) and posterior (28 points) chest walls. Additional electrodes were placed in order to record the standard 12-ECG and the Frank lead vectorcardiogram at the same time. Each electrode was connected to one input of a differential amplifier (x 1000), and the output from these amplifiers was digitized on-line at a sampling rate of 250 per second. The output of each amplifier was the potential recorded at each electrode position relative to that of the

### TABLE I TREADMILL EXERCISE PROTOCOL*

<table>
<thead>
<tr>
<th>Stage</th>
<th>Speed (mph)</th>
<th>Grade (%)</th>
<th>Time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.7</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
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</tr>
<tr>
<td>4</td>
<td>2.5</td>
<td>12</td>
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</tr>
<tr>
<td>5</td>
<td>3.4</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>5'</td>
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<td>3</td>
</tr>
<tr>
<td>6</td>
<td>4.2</td>
<td>16</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>5.0</td>
<td>18</td>
<td>3</td>
</tr>
</tbody>
</table>

*Sheffield protocol modified by the insertion of an intermediate stage (stage 5').

Fig.1. This map shows the area of ST-depression. Locations of the 6 standard precordial electrode sites (closed circles) are identified. A shadowed area corresponds to an area of ST depression greater than 0.1 mV. Four out of 87 pairs of wave forms obtained before (upper) and after (lower) treadmill exercise (Case 9) were displayed. Vertical lead points of A, E and I are on the right midaxillary, midsternal and left midaxillary lines, respectively. E4 and E6 are on the level of 5th and 2nd intercostal spaces, respectively. Exercise-induced ST depression is visible over the left anterior chest leads. On the other hand, this patient belongs to the inferior asynergy group and exercise-induced ST elevation is observed in inferior leads of the back. The method of the construction of this map was described in the text.

### TABLE II ARTERIOGRAPHIC SCORING SYSTEM*

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria for judging</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>1</td>
<td>Irregular arterial contour without a definite degree of stenosis.</td>
</tr>
<tr>
<td>2</td>
<td>Nonsignificant stenosis: &lt; 40% short, &lt; 20% long, &lt; 10% tubular.</td>
</tr>
<tr>
<td>3</td>
<td>Significant stenosis: 40 ~ 85% short, 20 ~ 60% long, 10 ~ 50% tubular.</td>
</tr>
<tr>
<td>4</td>
<td>Subocclusion: &gt; 85% short, &gt; 60% long, &gt; 50% tubular.</td>
</tr>
<tr>
<td>5</td>
<td>Occlusion</td>
</tr>
</tbody>
</table>

*From Pujadas and other authors.
short = stenosis shorter than 5 mm, long = stenosis between 5 and 10 mm, tubular = stenosis longer than 10 mm.
Wilson central terminal. On recording, all the
data were recorded from 400 msec before to 600
msec after the trigger site in the QRS complex of
the lead II ECG. Digital data were copied on a
digital cassette tape. Post-exercise data were
recorded one and a half min after the end of
the exercise. In order to minimize the effect of
respiration or heart position on the body surface
maps data sampling was always done at the
resting respiratory level in the supine position.

The flat portion of the PQ segment was
selected as a baseline or zero potential line. The
ST segment was analyzed at 60 msec after the J
point which was defined in the spatial vector.
For the construction of a body surface map
which expressed ST depression, the potential
difference between the pre-exercise and post-
exercise ST levels at each lead point was used.
In this map a negative area corresponds to an
area of ST depression. Depression of the ST level
greater than 0.1 mV was considered significant,
and inside the area of significant ST depression
isopotential lines at 0.1 mV intervals were
depicted using a minicomputer system which was
specifically developed for this purpose (Fig. 1).
Furthermore, the dimension (cm²) of the area of
ST depression (>0.1 mV) was automatically
calculated. This dimension was simply named
'area of ST depression'. For this purpose, the
circumference of the chest was normalized at 80
cm. The cassette tape copied by the HPM-5100
Heart Potential Mapper was used as the input to
this minicomputer system. Exercise-induced ST
elevation was considered to be present if the J
point was elevated greater than 1 mm compared
with the pre-exercise level, with a horizontal or
upsloping ST segment lasting more than 0.08 sec.

Selective coronary arteriographies were
performed in multiple projections by the Judkins
technique, and these arteriograms were evaluated
according to Pujadas without the knowledge of
the findings of the exercise test. The maximal
stenosis in each of the 4 main coronary stems
(right coronary artery, RCA; main trunk of the
left coronary artery, LMT; left anterior descending
artery, LAD; and left circumflex artery, LCX) was graded as follows:

Grade 0: Absolute arteriographic normality
of the arterial lumen.
Grade 1: Slight irregularities in the arterial
lumen but no localized stenosis.
Grade 2: Localized stenosis which does not
hinder the maximal coronary flow.
Grade 3: Stenosis which compromises the
maximal coronary flow significantly but does not hinder the rest
flow.

Grade 4: A subocclusion which inhibits even
the rest flow.
Grade 5: Total obstruction of the vessel with
no distal flow, except by collateral
circulation.

These grades were assigned according to Table II
which was made from the data obtained from
Pujadas and other authors. Concerning the
number of diseased vessels, Grades 3, 4 and 5
were considered significant stenosis, and LMT
disease was identified as a two-vessel disease with
LAD and LCX lesions. \( \Sigma \) Grade, as an index of
the severity of coronary artery disease, was
developed by adding up the grade numbers of all
4 main stems.

The left ventriculographies were performed in
the 30-degree right anterior and 60-degree left
anterior oblique views and these ventriculograms
were assessed visually by an experienced cardio-
vascular radiologist. Left ventricular asynergy
was defined as an akinetic or dyskinetic wall
motion.

The correlation between the ST-depression
area and \( \Sigma \) Grade was assessed using the least-
square method of linear regression analysis to
determine the correlation coefficient. Statistical
comparisons were done by the unpaired \( t \) test;
p <0.05 was considered significant. Quantitative
data are expressed as mean ± SEM.

RESULTS

Exercise tests and angiographic studies were
performed on all 27 patients. They were all males
and their age range was 37–68 years with a mean of
54.7. Clinical, angiographic and exercise test
data of these patients are presented in Table III.
Twelve normal subjects also underwent satisfac-
tory exercise tests. They were all males and their
mean age was 36 years (range 26–52). The ST-
depression area of these normal subjects ranged
from 0 to 111 cm² (45 ± 11 cm², Fig. 2). The
peak heart rate observed in these 12 normal
subjects during treadmill exercise was 130–188
beats/min (158 ± 4 beats/min).

These patients were divided into 3 groups on
the basis of the findings of the left ventriculo-
grams: the anterior asynergy group consisted of
6 patients who had asynergy at the anterior or
septal wall; the inferior asynergy group consisted of
6 patients who had asynergy at the inferior or

### TABLE III  CLINICAL, ANGIOGRAPHIC AND EXERCISE TEST DATA OF 27 PATIENTS

<table>
<thead>
<tr>
<th>Patients</th>
<th>Age (years)</th>
<th>Peak HR (beats/min)</th>
<th>Grade of four main coronary stems</th>
<th>ΣGrade</th>
<th>No. of significantly diseased arteries</th>
<th>ST-depression area (cm²)</th>
<th>Pathologic Q waves* in the left anterior chest leads</th>
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<td>LAD</td>
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</tr>
</tbody>
</table>

*Pathologic Q waves were defined as Q or QS waves ≥ 0.04 sec.

#Though this patient had no RCA lesion, he had an inferior asynergy for the reason that his LAD (grade 4) was long and reached the inferodiaphragmatic region of the left ventricle, and the posterior descending artery took off from the LCX (grade 4, left predominant pattern).

@Case 14 had proximal LAD obstruction, while Cases 16 and 17 had distal LCX obstruction. Their coronary arteriograms revealed abundant collateral circulation distal to the obstruction (Case 14, LCX and RCA → LAD; Case 16, RCA → LCX; Case 17, LCX and LAD → LCX). These collaterals were considered to protect the left ventricle from showing asynergic wall motion that might be caused by obstruction of the artery toward which the collateral circulation traveled.

Abbreviations: RCA = right coronary artery; LMT = left main trunk; LAD = left anterior descending artery; LCX = left circumflex artery.
posterior wall, and the no asynergy group consisted of 15 patients. Among these groups the difference in age, peak HR in exercise, \( \Sigma \) Grade or the number of significantly stenotic arteries was not statistically significant (Table IV). Of 6 patients in the anterior asynergy group, 5 (83%) had pathologic Q waves (pathologic Q waves were defined as QS or Q waves \( \geq 0.04 \) sec in the left anterior chest leads. These pathologic Q waves were also seen in some of the leads V2–V6 of the standard 12-ECG.

In patients with inferior asynergy the ST-depression area was 404 ± 68 cm\(^2\), which was larger than the 262 ± 69 cm\(^2\) of the no asynergy group, but the difference was not significant (Fig. 2). Both groups had significantly larger ST-depression areas compared with normal subjects (p < 0.001 for the inferior asynergy group, p < 0.02 for the no asynergy group). On the other hand, none of the 6 patients with anterior asynergy had an ST-depression area (Table IV).

Figure 3 shows the relationship of the ST-depression area to the severity of coronary artery disease (\( \Sigma \) Grade). Patients who belong to the inferior asynergy and no asynergy groups had ST-depression areas in proportion to their \( \Sigma \) Grade (r = 0.845, p < 0.001), whereas none of the patients with anterior asynergy had ST depression areas. In the inferior asynergy and no asynergy groups, patients whose \( \Sigma \) Grade was equal to or greater than 6 had ST depression areas greater than 250 cm\(^2\).

Of 15 patients with no asynergy, 10 had one-vessel disease, 3 had two-vessel and 2 had three-vessel disease (Table III). Of the 5 patients with no asynergy and multivessel disease (two- or three-vessel disease), all had ST-depression areas greater than 300 cm\(^2\), while none of the 10 patients who had no asynergy and one-vessel disease had such an ST-depression area. In both anterior and inferior asynergy groups, 3 had multivessel disease out of the 6 patients (Table III). None of the patients with anterior asynergy had ST-depression areas regardless of their number of diseased vessels. In inferior asynergy group, all patients with multivessel disease had

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**TABLE IV PATIENT CHARACTERISTICS**

<table>
<thead>
<tr>
<th></th>
<th>Anterior Asynergy (Group 1, n = 6)</th>
<th>Inferior Asynergy (Group 2, n = 6)</th>
<th>No Asynergy (Group 3, n = 15)</th>
<th>1 vs 2</th>
<th>1 vs 3</th>
<th>2 vs 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td>52.5 ± 2.6</td>
<td>55.7 ± 1.9</td>
<td>55.3 ± 2.5</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Peak HR (beats/min)</strong></td>
<td>137 ± 8</td>
<td>145 ± 11</td>
<td>131 ± 5</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td><strong>( \Sigma ) Grade</strong></td>
<td>8.0 ± 1.7</td>
<td>6.5 ± 0.9</td>
<td>6.1 ± 0.8</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td><strong>No. of significantly diseased arteries</strong></td>
<td>1.8 ± 0.4</td>
<td>1.5 ± 0.2</td>
<td>1.5 ± 0.2</td>
<td>NS</td>
<td>NS</td>
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</tr>
<tr>
<td><strong>ST-depression area (cm(^2))</strong></td>
<td>0</td>
<td>404 ± 68</td>
<td>262 ± 69</td>
<td>*</td>
<td>*</td>
<td>NS</td>
</tr>
</tbody>
</table>

*The difference in the ST-depression area between the anterior asynergy group and other groups was obvious although it could not be evaluated by the t test.*

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ST-depression areas greater than 300 cm². On the other hand, unlike no asynergy group, 2 of the 3 patients with one-vessel disease had also large (> 300 cm²) ST-depression areas.

Exercise-induced ST elevation was present in 83% (5/6) of the patients with anterior asynergy and 50% (3/6) of those with inferior asynergy. However, none (0/15) of the patients with no asynergy showed exercise-induced ST elevation.

**DISCUSSION**

In order to predict the severity of coronary artery disease quantitatively by the treadmill exercise test, many authors have attempted to make use of several hemodynamic responses instead of using only an ST depression. But the importance of ST depression as an indicator of myocardial ischemia is unchanged. In this report using body surface mapping we gave our attention to ST depression only and tried to assess the clinical significance of ST-depression areas in order to make a quantitative diagnosis of coronary artery disease.

This study revealed that, in patients with inferior asynergy and no asynergy of the left ventricle, the ST-depression area was proportional to the severity of the coronary artery disease (ΣGrade), whereas none of the patients with anterior asynergy had ST-depression areas regardless of their ΣGrade. Of 6 patients with anterior asynergy, 5 (83%) had pathologic Q waves in the left anterior chest leads, but none of the other groups had pathologic Q waves in the same region. Therefore, in patients without pathologic Q waves in the left anterior chest leads the severity of coronary artery disease could be predicted by the size of the ST-depression area (except one patient with anterior asynergy without pathologic Q waves, Case 3).

In patients who had a normal resting ECG, using a 14-lead ECG system (standard 12-ECG, excluding aVR, and leads CC5, CM5 and CL), reported that the extent of coronary artery disease could not be predicted accurately from the sum of ST-depression in 14 leads although the latter roughly corresponded to the number of vessels with stenosis ≥ 70%. Though they used a 14 lead system, it seems to be far from perfect for detecting precisely the changes in cardiac electrical activity induced by treadmill exercise. On the other hand, our system is considered to be able to detect the total body surface potentials using 87 leads distributed over not only the left anterior chest but also the right anterior chest and the back. For this reason, we believe, the ST-depression area computed in our system could be a useful indicator to predict the severity of coronary artery disease.

All of the patients (n = 5) with anterior asynergy and pathologic Q waves in the left anterior chest leads showed exercise-induced ST elevation at the corresponding region of the pathologic Q waves. Exercise-induced ST elevation has been attributed to abnormality of left ventricular wall motion. In the anterior asynergy group, the correlation between the ST-depression area and the severity of coronary artery disease seems to become less apparent by the exercise-induced ST elevation observed over the left anterior region of pathologic Q waves. It seems that reciprocal ST depression which might be observed elsewhere could hardly reach a significant level (> 0.1 mV), if any. It is supposed that additional RCA or LCX narrowing may affect the ST-depression area in patients with anterior asynergy. In the present study, we could not show the contribution of narrowings of these vessels to ST-depression area in anterior asynergy group because none of the patients who had RCA and/or LCX lesions in addition to LAD lesion had ST-depression areas. To determine the influence of RCA or LCX narrowing to ST-depression area in patients with anterior asynergy, further study is required.

On the other hand, inferior asynergy may...
enlarge the ST-depression area as a result of the reciprocal effect of ST elevation, which was observed in 3 (50%) patients in lower chest leads of our system (Cases 9–11, Fig. 1). In fact the ST-depression area of the inferior asynergy group was larger than that of the no asynergy group (404 ± 68 vs 262 ± 69 cm²), although the difference was not significant.

In patients with no asynergy, we showed that the ST-depression area was large (> 300 cm²) when the patient had a multivessel disease (Cases 13–17, Table III). Of patients with no asynergy and multivessel disease (n = 5, Cases 13–17), all had significant left coronary stenosis (Grade of LAD and/or LCX ≥ 4) and 4 (80%, Cases 13, 14, 16 and 17) had significant RCA stenosis (Grade of RCA ≥ 3). This result indicated that not only left coronary but also right coronary stenosis play an important role in producing the ST-depression area. In the no asynergy group, the ratio of ischemic muscle to total heart muscle is considered to be greater in patients with multivessel disease than those with one-vessel disease. This suggests that the ST-depression area of the no asynergy group reflects the amount of ischemic heart muscle caused by treadmill exercise.

Three of 6 patients with no asynergy and an ST-depression area > 300 cm² (Cases 14, 16 and 17) had an obstruction of one major coronary stem, and their coronary angiograms revealed abundant collateral circulation to the distal part of the obstruction (Case 14, LCX and RCA → LAD; Case 16, RCA → LCX; Case 17, LCX and LAD → LCX). Although these collaterals protected the left ventricle from showing asynergic wall motion, the myocardial distal to the obstruction was considered to be severely ischemic after treadmill exercise. This also suggests that in patients with no asynergy the ST-depression area and the amount of ischemic heart muscle correlates to each other.

Because the number of patients in this study was small we treated the inferior asynergy group and no asynergy group together in evaluating the correlation between the ST-depression area and ΣGrade. In the inferior asynergy group, the reciprocal effect of ST elevation may influence the ST-depression area, while this effect can probably be ignored in the no asynergy group. The clinical significance of the ST-depression area in the no asynergy group and the inferior asynergy group needs further investigation. In addition, the effect of right ventricular abnormal wall motion, which was not evaluated in the present study, on exercise-induced ST changes must be examined.

In conclusion this study emphasized the clinical significance of the ST-depression area for the quantitative and non-invasive diagnosis of coronary artery disease, especially in patients without pathologic Q waves in the left anterior chest leads. Furthermore, the ST-depression area computed in our system may prove a useful, non-invasive means for the assessment of medical and surgical treatment for patients with coronary artery disease.

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